America's Children and the Environment, Third Edition

DRAFT Indicators

Environments and Contaminants: Hazardous Air Pollutants

EPA is preparing the third edition of *America's Children and the Environment* (ACE3), following the previous editions published in December 2000 and February 2003. ACE is EPA's compilation of children's environmental health indicators and related information, drawing on the best national data sources available for characterizing important aspects of the relationship between environmental contaminants and children's health. ACE includes four sections: Environments and Contaminants, Biomonitoring, Health, and Special Features.

EPA has prepared draft indicator documents for ACE3 representing 23 children's environmental health topics and presenting a total of 42 proposed children's environmental health indicators. This document presents the draft text, indicator, and documentation for the hazardous air pollutants topic in the Environments and Contaminants section.

THIS INFORMATION IS DISTRIBUTED SOLELY FOR THE PURPOSE OF PRE-DISSEMINATION PEER REVIEW UNDER APPLICABLE INFORMATION QUALITY GUIDELINES. IT HAS NOT BEEN FORMALLY DISSEMINATED BY EPA. IT DOES NOT REPRESENT AND SHOULD NOT BE CONSTRUED TO REPRESENT ANY AGENCY DETERMINATION OR POLICY.

For more information on America's Children and the Environment, please visit <u>www.epa.gov/ace</u>. For instructions on how to submit comments on the draft ACE3 indicators, please visit <u>www.epa.gov/ace/ace3drafts/</u>.

1 Hazardous Air Pollutants

Hazardous Air Pollutants (HAPs) are air pollutants that are known or suspected to cause serious
human health effects or adverse environmental effects.¹ Health effects associated with HAPs
include cancer, asthma and other respiratory ailments, birth defects, reproductive effects, and
neurological problems such as learning disabilities and hyperactivity.²⁻¹⁵ The Clean Air Act
identifies 187 substances as HAPs. Examples include benzene, trichloroethylene, mercury,
chromium, and dioxin. The "criteria" air pollutants such as ozone and particulate matter are
excluded from the HAPs list.

- 9 HAPs are emitted from a diverse range of facilities, businesses, and vehicles that are grouped
- 10 into three general categories: major sources, area sources, and mobile sources. Major sources
- 11 typically are large industrial facilities such as chemical manufacturing plants, refineries, and
- 12 waste incinerators. These sources may release air toxics from equipment leaks, when materials
- 13 are transferred from one location to another, or during discharge through emission stacks or
- 14 vents. Area sources typically are smaller stationary facilities such as dry cleaners, auto body
- 15 repair shops and small manufacturing operations. Though emissions from individual area sources
- 16 often are relatively small, collectively they can be of concern—particularly where large numbers 17 of sources are located in heavily populated areas. Mobile sources include both on-road sources,
- of sources are located in heavily populated areas. Mobile sources include both on-road sources,such as cars, light trucks, large trucks, and buses, and non-road sources such as farm and
- 19 construction equipment, lawn and garden equipment, marine engines, aircraft, and locomotives.
- 20 Some HAPs are also emitted from natural sources such as volcanoes.
- 21 EPA relies on both monitoring and modeled data to characterize ambient air concentrations of
- HAPs, and to estimate potential human exposure and risk of adverse health effects associated
- 23 with these toxics. EPA and state monitoring programs currently do not adequately cover all the
- 24 places where people live in the United States. For this reason, the indicator presented here relies
- 25 on modeled data from the National Air Toxics Assessment.¹⁶ The indicator presents the
- 26 percentage of children living in counties with estimated HAP concentrations greater than
- 27 benchmark comparison levels derived from health effects information.

1 Indicator E4: Percentage of children ages 0 to 17 years living in

2 counties where estimated hazardous air pollutant

3 concentrations were greater than health benchmarks in 2002

Overview

Indicator E5 presents estimates of the percentage of children living in counties with hazardous air pollutant (HAP) concentrations greater than benchmarks representing levels of concern for health effects. The HAP concentrations are computer model estimates for 2002, representing all identified sources of HAP emissions, including factories and motor vehicles. The health benchmarks are based on concerns for cancer and other serious health effects that may be associated with HAP exposure.

4

5 National Air Toxics Assessment

6 EPA's National Air Toxics Assessment (NATA) provides estimated concentrations of 183 HAPs

7 in ambient air for the year 2002. NATA is the most comprehensive resource on potential human

8 exposure to and risk of adverse health effects from HAPs in the United States. Monitoring data

9 are insufficient to characterize HAP concentrations across the country because of the limited

10 number of monitors, and because concentrations of many HAPs may vary considerably within a

11 metropolitan area or region.

12 Under NATA, EPA develops modeled estimates of ambient concentrations of HAPs using

13 estimated emissions data from major, area, onroad mobile, and non-road mobile sources. These

emissions data are collected and updated periodically, and are maintained in an emissions

15 inventory. The original NATA was developed using emissions data for the year 1996. Since the

16 initial release, EPA has developed additional estimates of ambient air concentrations of HAPs

17 using updated emissions inventories for 1999 and 2002.

18 The most recent assessment developed estimated ambient concentrations of 183 air toxics for the

19 year 2002. A computer model provided estimates for every county in the United States. These

20 estimates generally are consistent with the limited set of ambient air toxics monitoring data,

21 although at many locations the model estimates for some HAPs are lower than measured

22 concentrations. The 2002 NATA estimates do not reflect any changes in emissions that may have

23 occurred since 2002 due to new regulations, new technologies, changes in economic activity, or

changes in the vehicle fleet and vehicle miles travelled.

25 Health Benchmarks for Hazardous Air Pollutants

- 26 The HAPs indicator shown here reflects comparisons of modeled concentrations of HAPs in
- ambient air for 2002 with three health benchmark concentrations derived from scientific
- assessments conducted by EPA and other environmental agencies.¹⁷⁻²¹

- 1 Two benchmarks reflect potential cancer risks, at levels of 1-in-100,000 risk and 1-in-10,000
- 2 risk. If a particular hazardous air pollutant is present in ambient air at a 1-in-100,000 benchmark
- 3 concentration, for example, one additional case of cancer would be expected in a population of
- 4 100,000 people exposed for a lifetime. The third benchmark concentration corresponds to the
- 5 level at which exposure to the hazardous air pollutant is estimated to be of minimal risk;
- 6 exposures above this benchmark may be associated with adverse health effects other than cancer,
- 7 such as respiratory or neurological effects.
- 8 The health benchmarks are generally derived from laboratory animal studies, although for some
- 9 HAPs they are derived from human epidemiological studies of workers exposed on the job. For
- 10 some HAPs, even the animal studies are very limited and no benchmark has been derived. Health
- benchmarks were available to assess 80 HAPs as cancer-causing agents and 104 HAPs as agents
- 12 that cause adverse health effects other than cancer. Some HAPs had benchmarks for both cancer
- 13 and non-cancer health endpoints. Therefore, cancer and non-cancer risks were estimated for a
- 14 total of 134 air toxics.
- 15 The three benchmarks generally reflect health risks to adults, rather than potential risks to
- 16 children or risks in adulthood stemming from childhood exposure. Further, the benchmarks
- 17 reflect risks of continuous exposure over the course of a lifetime. Potential risks from very high
- 18 short-term exposures, or from elevated exposures that may be experienced during childhood, are
- 19 not addressed by these benchmarks.

20 Data Presented in the Indicator

- 21 Indicator E5 presents the percentage of children living in counties where estimated 2002 HAP
- 22 concentrations exceeded benchmark levels for cancer (at levels of 1-in-100,000 risk and 1-in-
- 23 10,000 risk) and for other adverse health effects. The indicator is calculated by comparing the
- estimated HAP concentrations for each U.S. county in 2002 to each of the benchmark
- 25 concentrations.
- 26 The indicator presents results only for 2002, and does not compare results across assessment
- 27 years, such as between 1999 and 2002, because each update of the assessment brings new
- 28 improvements to methods. For example, improvements to the emissions estimation
- 29 methodologies made in the 2002 assessment were not applied to the earlier versions, so the
- 30 ambient concentration estimates are not entirely comparable between years.
- 31 Actual exposures may differ from ambient concentrations. Indoor concentrations of HAPs from
- 32 outdoor sources may be slightly lower than ambient concentrations, although they can be
- 33 significantly higher if any indoor sources are present. Levels of some hazardous pollutants may
- 34 be substantially higher inside cars and school buses, and those higher levels would increase the
- 35 risks.
- 36 In addition, this indicator only considers exposures to air toxics that occur by inhalation. For
- 37 many air toxics, dietary exposures are also important. Air toxics that are persistent in the
- 38 environment settle out of the atmosphere onto land and water, and then accumulate in fish and
- 39 other animals in the food web. For HAPs that are persistent in the environment and accumulate

- 1 significantly in food, exposures through food consumption typically are greater than inhalation
- 2 exposures. HAPs for which food chain exposures are important include mercury, dioxins, and
- 3 PCBs.²²⁻²⁴
- 4 The comparison of ambient HAP concentrations in 2002 to the health benchmarks is not
- 5 equivalent to an estimate of risk to the population from chronic HAP exposure. Actual risks to
- 6 health depend on concentrations of HAPs in many environments over an extended period of
- 7 time. Ambient concentrations will change over time as the mix of sources changes (e.g., due to
- 8 businesses opening and closing), vehicle use changes (e.g., more cars and trucks traveling longer
- 9 distances), and regulatory controls are applied. In addition, children spend most of their time 10 indoors at home, at school, or at child care centers, and pollutant concentrations in indoor
- environments may be greater than or lesser than the modeled ambient concentrations.
- 12 In addition to the indicator presented here, which is based on where children live, the same
- 13 statistics are calculated based on where children's schools are located (see data tables).
- 14 Exposures at school are an important consideration, as children spend an average of 33 hours per
- 15 week in school.²⁵



• In 2002, all children lived in counties in which HAPs concentrations combined to exceed the 1-in-100,000 cancer risk benchmark.

1

2

3

4 5

6

7

8

9

- About 6% of children lived in counties in which HAPs combined to exceed the 1-in-10,000 cancer risk benchmark. The pollutants that contributed most to this result were hydrazine, chromium compounds, benzene, and carbon tetrachloride. Hexavalent chromium and benzene are considered by EPA to be "known human carcinogens," and hydrazine and carbon tetrachloride are "probable human carcinogens."
- Approximately 85% of children lived in counties in which at least one HAP exceeded the
 benchmark for health effects other than cancer. In almost all cases, this result was attributable
 to the pollutant acrolein, which is a respiratory irritant. More than 90% of acrolein emissions
 are from wood-burning fires and mobile sources such as cars, trucks, buses, planes, and

- 1 construction equipment.
- Exposures to diesel particulate matter from diesel engine emissions are not included in this
 indicator due to uncertainty regarding the appropriate values to use as cancer benchmarks.
 Some studies have found that cancer risks from diesel particulate matter exceed those of the
 HAPs considered in this indicator.²⁶ Although EPA does not endorse any particular cancer
 benchmark value for diesel particulate matter, if the State of California's benchmark for
 diesel particulate matter were used in this analysis, 96% of children would live in counties
 where HAP estimates combined to exceed the 1-in-10,000 cancer risk benchmark.
- 10
- In 2002, all children's schools were located in census tracts where HAPs concentrations
 combined to exceed the 1-in-100,000 cancer risk benchmark. Approximately 5% of children
 attended schools in census tracts where the HAPs concentrations exceeded the higher 1-in 10,000 cancer risk benchmark.
- 15
- About 82% of children attended schools that were located in census tracts where at least one
 HAP exceeded the benchmark for health effects other than cancer.

Data Tables

Table E4: Percentage of children ages 0 to 17 years living in counties where estimated hazardous air pollutant concentrations were greater than health benchmarks in 2002

Health Benchmark	
Cancer, one in 100,000	100%
Cancer, one in 10,000	5.9%
Other health effects	85%

DATA: U.S. Environmental Protection Agency, National Air Toxics Assessment

Table E4a: Percentage of schoolchildren attending schools in census tracts where estimated hazardous air pollutant concentrations were greater than health benchmarks in 2002

Health Benchmark		
Cancer, one in 100,000	100%	
Cancer, one in 10,000	4.7%	
Other health effects	82%	

13

14 DATA: U.S. Environmental Protection Agency, National Air Toxics Assessment

15

6

References

1. U.S. Environmental Protection Agency. 2009. *About Air Toxics*. Retrieved August 6, 2009 from <u>www.epa.gov/ttn/atw/allabout.html</u>.

2. Delzell, E., N. Sathiakumar, M. Hovinga, M. Macaluso, J. Julian, R. Larson, P. Cole, and D.C. Muir. 1996. A follow-up study of synthetic rubber workers. *Toxicology* 113 (1-3):182-9.

3. Grandjean, P., E. Budtz-Jorgensen, R.F. White, P.J. Jorgensen, P. Weihe, F. Debes, and N. Keiding. 1999. Methylmercury exposure biomarkers as indicators of neurotoxicity in children aged 7 years. *American Journal of Epidemiology* 150 (3):301-5.

4. Jacobson, J.L., and S.W. Jacobson. 1997. Teratogen update: polychlorinated biphenyls. *Teratology* 55 (5):338-47.

5. Leikauf, G.D., S. Kline, R.E. Albert, C.S. Baxter, D.I. Bernstein, and C.R. Buncher. 1995. Evaluation of a possible association of urban air toxics and asthma. *Environmental Health Perspectives* 103 Suppl 6:253-71.

6. Marlowe, M., A. Cossairt, C. Moon, J. Errera, A. MacNeel, R. Peak, J. Ray, and C. Schroeder. 1985. Main and interaction effects of metallic toxins on classroom behavior. *Journal of Abnormal Child Psychology* 13 (2):185-98.

7. Matanoski, G.M., and L. Schwartz. 1987. Mortality of workers in styrene-butadiene polymer production. *Journal of Occupational and Environmental Medicine* 29 (8):675-80.

8. National Toxicology Program. 1993. *Toxicology and Carcinogenesis of 1,3-butadiene (CAS No. 106-99-0) in B6C3F1 Mice (Inhalation Studies)*. Research Triangle Park, NC: National Toxicology Program. NTP TR 434, NIH Pub. No. 93-3165.

9. Needleman, H.L., A. Schell, D. Bellinger, A. Leviton, and E.N. Allred. 1990. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *New England Journal of Medicine* 322 (2):83-8.

10. Perera, F.P., Z. Li, R. Whyatt, L. Hoepner, S. Wang, D. Camann, and V. Rauh. 2009. Prenatal Airborne Polycyclic Aromatic Hydrocarbon Exposure and Child IQ at Age 5 Years. *Pediatrics* 124 (2):e195-202.

11. Rinsky, R.A., A.B. Smith, R. Hornung, T.G. Filloon, R.J. Young, A.H. Okun, and P.J. Landrigan. 1987. Benzene and leukemia. An epidemiologic risk assessment. *New England Journal of Medicine* 316 (17):1044-50.

12. Rinsky, R.A., R.J. Young, and A.B. Smith. 1981. Leukemia in benzene workers. *American Journal of Industrial Medicine* 2 (3):217-45.

Rothman, N., G.L. Li, M. Dosemeci, W.E. Bechtold, G.E. Marti, Y.Z. Wang, M. Linet, L.Q. Xi, W. Lu, M.T.
 Smith, N. Titenko-Holland, L.P. Zhang, W. Blot, S.N. Yin, and R.B. Hayes. 1996. Hematotoxicity among Chinese
 workers heavily exposed to benzene. *American Journal of Industrial Medicine* 29 (3):236-46.

14. Ward, E., R. Hornung, J. Morris, R. Rinsky, D. Wild, W. Halperin, and W. Guthrie. 1996. Risk of low red or
white blood cell count related to estimated benzene exposure in a rubberworker cohort (1940-1975). *American Journal of Industrial Medicine* 29 (3):247-57.

15. Ware, J.H., J.D. Spengler, L.M. Neas, J.M. Samet, G.R. Wagner, D. Coultas, H. Ozkaynak, and M. Schwab.
 1993. Respiratory and irritant health effects of ambient volatile organic compounds. The Kanawha County Health Study. *American Journal of Epidemiology* 137 (12):1287-301.

16. U.S. Environmental Protection Agency. 2009. *National Air Toxics Assessment*. Office of Air Quality Planning
 and Standards. Retrieved August 6, 2009 from <u>www.epa.gov/ttn/atw/natamain</u>.

17. Agency for Toxic Substances and Disease Registry. 2009. *Minimal Risk Levels (MRLs) for Hazardous Substances*. Retrieved August 14, 2009 from <u>http://www.atsdr.cdc.gov/mrls/index.html</u>.

18. California Environmental Protection Agency. 2009. All OEHHA Acute, 8-hour and Chronic Reference Exposure Levels (chRELs) as on December 18, 2008. Retrieved August 14, 2009 from http://www.oehha.ca.gov/air/allrels.html.

19. U.S. Environmental Protection Agency. 1997. *Health Effects Assessment Summary Tables (HEAST)*. Washington, DC: U.S. Environmental Protection Agency. EPA-540-R-97-036.

20. U.S. Environmental Protection Agency. 2009. *Integrated Risk Information System*. Retrieved August 6, 2009 from <u>www.epa.gov/iris/index.html</u>.

21. U.S. Environmental Protection Agency. *Health Effects Information Used in Cancer and Noncancer Risk Characterization for the 2002 National-Scale Assessment*. Retrieved August 14, 2009 from http://www.epa.gov/ttn/atw/nata2002/02pdfs/health_effects.pdf.

22. National Research Council. 2000. *Toxicological Effects of Methylmercury*. Washington, DC: National Academy Press.

23. U.S. Environmental Protection Agency. 1996. *Mercury Study Report to Congress, Volumes I to VII*. Washington DC: Office of Air Quality Planning and Standards. <u>http://www.epa.gov/mercury/report.htm</u>.

24. U.S. Environmental Protection Agency. 2000. *Deposition of Air Pollutants to the Great Waters: Third Report to Congress*. Washington, DC. <u>http://epa.gov/ttncaaa1/t3/reports/head_2kf.pdf</u>.

25. U.S. Department of Education. 2010. Average number of hours and percentage of the student school week that public school teachers of first- through fourth-grade, self-contained classrooms spent on each of four subjects, total instruction hours per week on four subjects, total time spent delivering all instruction per week, and average length of student school week: Selected years 1987-88 through 2007-08. National Center for Education Statistics. Retrieved July 22, 2010 from http://nces.ed.gov/surveys/sass/tables/sass0708_005_t1n.asp.

26. South Coast Air Quality Management District. 2000. *Multiple Air Toxics Exposure Study II*. <u>http://www.aqmd.gov/matesiidf/matestoc.htm</u>.

1 Metadata

2

Metadata for	National Air Toxics Assessment (NATA)
Brief description of the	The National Air Toxics Assessment is EPA's ongoing
data set	comprehensive evaluation of air toxics in the United States.
	NATA provides estimates of the risk of cancer and other
	serious health effects from inhaling air toxics in order to
	inform both national and more localized efforts to identify
	and prioritize air toxics, emission source types, and locations
	that are of greatest potential concern in terms of contributing
	to population risk.
Who provides the data	U.S. Environmental Protection Agency, Office of Air Quality
set?	Planning and Standards.
How are the data	Emissions inventory data for individual HAPs are collected
gathered?	from data reported by large individual facilities (point
	sources) and estimated for area and mobile sources using
	various emissions inventory models. The compiled inventory
	is called the National Emissions Inventory. Ambient
	concentrations are estimated using an air dispersion model.
	Population exposures are estimated based on a screening-
	level inhalation exposure model.
What documentation is	See <u>http://www.epa.gov/nata2002</u> for detailed description of
available describing data	NATA organization and data collection practices.
collection procedures?	
What types of data	Modeled ambient concentrations, exposure concentrations,
relevant for children's	cancer risks, and non-cancer hazard indices for each HAP in
environmental health	each county and each census tract.
indicators are available	
from this database?	
What is the spatial	National.
representation of the	
database (national or	
other)?	
Are raw data (individual	Modeled ambient and exposure concentrations for each HAP
measurements or survey	in each county and census tract are available.
responses) available?	
How are database files	http://www.epa.gov/ttn/atw/nata2002/tables.html.
obtained?	
Are there any known	NATA results provide answers to questions about emissions,
data quality or data	ambient air concentrations, exposures and risks across broad
analysis concerns?	geographic areas (such as counties, states, and the nation) at a
	moment in time. These assessments are based on assumptions
	and methods that limit the range of questions that can be

DRAFT Indicator for Third Edition of America's Children and the EnvironmentPage 10February 2011DO NOT QUOTE OR CITE

Metadata for	National Air Toxics Assessment (NATA)
	answered reliably. The results cannot be used to identify
	exposures and risks for specific individuals, or even to
	identify exposures and risks in small geographic regions.
	These estimates reflect chronic exposures resulting from the
	inhalation of the air toxics emitted and do not consider
	exposures that may occur indoors or as a results of exposures
	other than inhalation (i.e., dermal or ingestion). Methods used
	in NATA were peer reviewed by EPA's Science Advisory
	Board; the SAB report is available at
	http://www.epa.gov/ttn/atw/sab/sabrept1201.pdf.
What documentation is	See http://www.epa.gov/nata2002.
available describing OA	
procedures?	
For what years are data	1996, 1999, 2002.
available?	
What is the frequency of	Approximately every three years.
data collection?	
What is the frequency of	Approximately every three years.
data release?	
Are the data comparable	Data for different NATA assessments are not comparable
across time and space?	across time due to improvements in the estimated national
	emissions inventory, increases in the numbers of modeled
	HAPs, and improvements in the health data information. Data
	may not be comparable over space due to quality differences
	in emissions inventory reporting.
Can the data be stratified	Data can be stratified by state, county, and census tract.
by race/ethnicity,	
income, and location	
(region, state, county or	
other geographic unit)?	

1

2 Methods

34 Indicator

E4. Percentage of children ages 0 to 17 years living in counties where estimated hazardous air pollutant concentrations were greater than health benchmarks in 2002.

89 Summary

10

5 6

7

11 EPA's Office of Air Quality Planning and Standards (OAQPS) estimated county annual average outdoor concentrations of 183 hazardous air pollutants (HAPs), also known as air toxics, as part 12 13 of EPA's National Air Toxics Assessment (NATA) for the calendar year 2002. EPA used a computer dispersion model, the Assessment System for Population Exposure Nationwide 14 15 (ASPEN), to estimate these concentrations based on the 2002 emissions inventory of air toxics 16 emissions from outdoor sources. The lifetime cancer risks posed by HAPs in each county were calculated by multiplying the ambient concentration of each HAP by the inhalation unit risk 17 18 estimate (URE) of that HAP. The risk estimates for all modeled HAPs with cancer unit risk 19 estimates then were summed together to provide a combined cancer risk estimate. The counties 20 for which this value exceeded 1-in-100,000 and 1-in-10,000 were identified, producing two lists 21 of counties. For each list of counties, the number of children ages 0 to 17 years in the identified 22 counties was summed together. The resulting value then was divided by the number of children 23 ages 0 to 17 years in all counties in the United States, yielding the percentage of children living 24 in counties where the concentrations of carcinogenic hazardous air pollutants exceeded the two 25 benchmark cancer risk levels. For non-cancer health benchmarks, counties in which the annual 26 average concentration exceeded the reference concentration for any HAP were identified. The 27 number of children ages 0 to 17 years in the identified counties was summed together. The 28 resulting value was then divided by the number of children ages 0 to 17 years in all counties in the United States to yield the percentage of children living in counties where the concentration of 29 30 one or more hazardous air pollutants exceeded the health benchmark for effects other than 31 cancer. Table E4a provides the same set of results for the percentages of schoolchildren 32 attending public or private elementary or secondary schools in census tracts where the 33 concentrations of carcinogenic hazardous air pollutants exceeded the two benchmark cancer risk 34 levels or where the concentration of one or more hazardous air pollutants exceeded the health benchmark for effects other than cancer. 35 36 37 **Overview of Data Files** 38 39 The following files are needed to calculate this indicator:

- 40
- County annual averages. There is one ACCESS data table for each of the modeled HAPs.
 This table contains the state and county FIPS codes, the total annual average ASPEN modeled concentration, and other information not used for these calculations. These
 ACCESS files were altained from the NATA 2002 Web received
- 44 ACCESS files were obtained from the NATA 2002 Web page:

1 2 2	http://www.epa.gov/ttn/atw/nata2002/tables.html. See under "2002 County-Level Modeled Ambient Concentrations, Exposures, and Risks."
3 4	For the eight individual air toxics groups of Polycyclic Organic Matter (POMs) and for
5	two Polycyclic Aromatic Hydrocarbons (PAHs), 1-methylnaphthalene, and 2-
0 7	over these 10 hazardous air pollutants. We obtained the county appual average files for
8	these 10 individual hazardous air pollutants directly from EPA OAOPS ¹
9	
10	• Health effects criteria. This file health effects.pdf lists the cancer unit risk estimate
11	(URE) for all carcinogenic HAPs and lists the reference concentrations (RfC) for HAPs
12	with non-cancer health effects. We obtained this file from the Web page:
13	http://www.epa.gov/ttn/atw/nata2002/riskbg.html. See under "Health Effects Criteria
14	(PDF)."
15	
16 17	• Census data. This file contains the state and county FIPS codes, year, and children's nonverticen For 2002, we obtained this information from the bridged race Vintege 2007.
1/ 18	population. For 2002, we obtained this information from the offdged-face vintage 2007
19	posteensal population me.
20	National Center for Health Statistics. Postcensal estimates of the resident
21	population of the United States for July 1, 2000-July 1, 2007, by year, county,
22	age, bridged race, Hispanic origin, and sex (Vintage 2007). Prepared under a
23	collaborative arrangement with the U.S. Census Bureau; released August 7, 2008.
24	Available from: <u>http://www.cdc.gov/nchs/nvss/bridged_race.htm</u> as of September
25	5, 2008.
26	The menulations have and example shows altering diversity and the second diverse of the second of the
21	The populations by year and county were obtained by summing across the ages 0 to 17
20 29	years merusive.
30	Air Quality Data
31	
32	Health effects criteria for the hazardous air pollutants studied in the National Air Toxics
33	Assessment (NATA) for 2002 were obtained from the file health_effects.pdf on the Web page:
34	http://www.epa.gov/ttn/atw/nata2002/riskbg.html. See under "Health Effects Criteria (PDF)."
35	This file includes the cancer unit risk estimates (URE) and non-cancer reference concentrations
36	(RfC) for the 183 HAPs modeled in NATA 2002. Some of the HAPs had no URE reported, and
3/ 20	so were treated as having no cancer risk. Some of the HAPs had no RIC reported, and so were
20 20	reported LIRE nor a reported RfC From discussions with EPA OAOPS staff we discovered that
40	the reported URE value for acetaldehyde had been rounded in that file. We used the original
41	value $0.0000022 (\text{ug/m}^3)^{-1}$ for these analyses.
42	

¹ Ted Palma, EPA OAQPS, <u>palma.ted@epa.gov</u>, 919-541-5470.

1 2 3 4	Estimated county average annual outdoor concentrations for the year 2002 were obtained from the Web page: <u>http://www.epa.gov/ttn/atw/nata2002/tables.html</u> . See under "2002 County-Level Modeled Ambient Concentrations, Exposures, and Risks."
5 6 7 8 9	We obtained ACCESS files with ASPEN estimated concentrations for each of 175 HAPs. One of these HAPs was named "PAHPOM." This denotes the total concentration summed over the eight individual air toxics groups of Polycyclic Organic Matter (POMs) and two Polycyclic Aromatic Hydrocarbons (PAHs), 1-methylnaphthalene, and 2-chloroacetophenone. In order to apply appropriate UREs and RFCs to these 10 individual HAPs, we obtained county average
10	concentration files in ACCESS format for these 10 individual HAPs directly from EPA
11	OAQPS. ² For 2-chloroacetophenone, we used the file directly supplied by EPA OAQPS instead
12	of the file on the website, which had very similar, but not identical county concentration values.
13	Therefore we analyzed 183 individual HAPs. The named HAP from each of these 183 county
14	average concentration files matched exactly one of the HAPs listed in the health_effects.pdf file,
15	with three exceptions: The HAP "1,2,3,4,5,6-HEXACHLOROCYCLYHEXANE (ALL
10	STEREO ISOMERS) was matched to the risks for "Lindane (all isomers)." The HAP "I-
1/ 10	methylnaphinalene was assumed to have no cancer and no non-cancer health risks, based on a
10	for "Chromium VI compounds"
19 20	for Chromium vi compounds.
20	Census Data
$\frac{21}{22}$	Census Data
23	We obtained children's populations by county for the year 2002. The source was the bridged-
24	race Vintage 2007 postcensal population file:
25	
26	National Center for Health Statistics. Postcensal estimates of the resident
27	population of the United States for July 1, 2000-July 1, 2007, by year, county,
28	age, bridged race, Hispanic origin, and sex (Vintage 2007). Prepared under a
29	collaborative arrangement with the U.S. Census Bureau; released August 7, 2008.
30	Available from: <u>http://www.cdc.gov/nchs/nvss/bridged_race.htm</u> as of September
31	5, 2008.
32	
33 34 35 36	This file gives county populations by age and sex for 2000 to 2007. We summed these populations for 2002 by county across all ages 17 years and under, all races and ethnicities, and both sexes.
37	The NATA 2002 modeling used year 2000 census boundaries and therefore excluded the new
38	Broomfield, Colorado county which was created in 2001 from portions of Adams, Boulder,
39	Jefferson, and Weld counties. The 2007 postcensal population file included this county for the
40	year 2002, with a children's population of 12,169. To more accurately account for these 12,169
41	children, they were reallocated to Adams, Boulder, Jefferson, and Weld counties in proportion to
42	the children's populations of those four counties.
43	
44	

² Ted Palma, EPA OAQPS, <u>palma.ted@epa.gov</u>, 919-541-5470.

1 2	Calculation of Indicator
2 3 1	Indicator E4 is calculated as follows.
5 6 7 8 9	1. For each county, the cancer risk for each carcinogenic HAP is estimated by multiplying the ASPEN estimated annual average outdoor concentration ($\mu g/m^3$) by the unit risk estimate, URE. The URE is an estimate of the excess cancer risk resulting from a lifetime of continuous exposure to a pollutant at a concentration of one microgram per cubic meter (1 $\mu g/m^3$) in air. Thus,
11 12 13	Cancer risk (county C, HAP H) = ASPEN Annual Concentration (county C, HAP H) × URE (HAP H)
14 15 16	2. The total cancer risk for each county is estimated by summing the cancer risks across all carcinogenic HAPs:
17 18	Total cancer risk (county C) = Σ Cancer risk (county C, HAP H)
19 20	where this sum is across all HAPs.
21 22 23	3. The set of counties with a total cancer risk greater than 1-in-100,000 was listed. These are the affected counties.
24 25	4. The total population of children 0-17 living in the step 3 counties was summed:
26 27	Population affected = Σ Pop (county C), summed over the affected counties only
28 29	5. The total U.S. population of children 0-17 was summed:
30 31	U.S. Population = Σ Pop (county C), summed over all counties in the United States
32 33 34	6. The percentage of affected children was calculated by dividing the population affected by the total U.S. population:
35 36	Percentage children affected = [Population affected / U.S. Population] \times 100%
37 38 39	The percentage affected in step 6 is the percentage of children exceeding the 1-in-100,000 cancer health benchmark.
40 41 42 43	A very similar calculation gives the percentage of children exceeding the 1-in-10,000 cancer health benchmark. The only change is to redefine the list of affected counties in step 6 as those counties exceeding the 1-in-10,000 cancer risk.
44 45	The calculation for the other health effects benchmark proceeds as follows.

1 2 3	1. For each HAP with a reference concentration, we list the counties affected by non-cancer effects from that HAP. A county is affected by a given HAP if the ASPEN estimated annual average outdoor concentration exceeds the reference concentration.
4 5 6	2. We list the affected counties as any county that is affected by non-cancer effects from one or more of the modeled HAPs.
7 8	Now repeat the cancer risk steps 4, 5, and 6 above using the new list of affected counties. The
9 10	percentage affected is the percentage of children exceeding the non-cancel health benchmark.
11	Children at Schools
12	
13 14	Table E4a provides the percentages of schoolchildren attending public or private elementary or secondary schools in census tracts where the ambient concentrations of carcinogenic hazardous
15 16	air pollutants exceeded the two benchmark cancer risk levels or where the concentration of one or more hazardous air pollutants exceeded the health benchmark for effects other than cancer.
17	
18	The schools data used for these calculations were obtained by EPA ⁵ from the U.S. Department of Education. Data on public schools for the school year 2006, 2007 were obtained from the Dublic
20	Elementary/Secondary School Universe Survey Data Data on private schools for the school year
21	2005-2006 were obtained from the Private School Universe Survey:
22	
23 24 25	• Public Elementary/Secondary School Universe Survey Data. EPA selected data for the school year 2006-2007 and all public elementary and secondary schools in the database. Selected variables used for these analyses were: total students, school location latitude
26 27	and longitude.
28 29	These data were obtained from the following website:
30 31	http://nces.ed.gov/ccd/
32 33 34	• Private School Universe Survey Data. EPA selected data for the school year 2005-2006 and all private elementary and secondary schools in the database. Selected variables used for these analyses were: total students, school location latitude and longitude.
35 36 37	These data were obtained from the following website:
38 39	http://nces.ed.gov/surveys/pss/pssdata.asp
40 41	This analysis also used the NATA 2002 tract annual averages files:
42 43	• Tract annual averages. There is one ACCESS data table for each of the modeled HAPs. This table contains the state, county, and tract FIPS codes, the total annual average

³ Mark Morris, EPA OAQPS. (919) 541-5416. <u>morris.mark@epa.gov</u>

1 2 3 4 5	ASPEN-modeled concentration, and other information not used for these calculations These ACCESS files were obtained from the NATA 2002 Web page: <u>http://www.epa.gov/ttn/atw/nata2002/tables.html</u> . See under "2002 Tract-Level Modeled Ambient Concentrations, Exposures and Risks."
6 7 8 9 10	For the eight individual air toxics groups of Polycyclic Organic Matter (POMs) and for two Polycyclic Aromatic Hydrocarbons (PAHs), 1-methylnaphthalene, and 2-chloroacetophenone, the Web page provides a file with the total concentrations summed over these 10 hazardous air pollutants. We obtained the tract annual average files for these ten individual hazardous air pollutants directly from EPA OAQPS ⁴ .
11 12 13	Public Elementary/Secondary School Universe Survey Data.
13 14 15 16 17 18 19 20 21	The Common Core of Data (CCD) is a program of the U.S. Department of Education's National Center for Education Statistics that annually collects fiscal and non-fiscal data about all public schools, public school districts, and state education agencies in the United States. The data are supplied by state education agency officials and include information that describes schools and school districts, including name, address, and phone number; descriptive information about students and staff, including demographics; and fiscal data, including revenues and current expenditures.
21 22 23	Private School Universe Survey.
25 24 25 26 27 28 29 30 31 32 33 34 35	In 1988, the National Center for Education Statistics (NCES) introduced a proposal to develop a private school data collection that would improve on the sporadic collection of private school data dating back to 1890 and improve on commercially available private school sampling frames. Since 1989, the U.S. Bureau of the Census has conducted the biennial Private School Universe Survey (PSS) for NCES. The PSS is designed to generate biennial data on the total number of private schools, students, and teachers, and to build a universe of private schools in the 50 states and the District of Columbia to serve as a sampling frame of private schools for NCES sample surveys. The target population for the PSS is all schools in the 50 states and the District of Columbia that are not supported primarily by public funds, provide classroom instruction for one or more of grades kindergarten through 12 (or comparable ungraded levels), and have one or more teachers.
36 37 38 39 40 41 42 43 44	Calculation of Indicator EPA compiled the public and private school data into a single database. For each school, the latitude and longitude were used to calculate the BLOCK_ID, a 15 character identifier for the census block nearest to the school, as defined by the distance between the school and the census block centroid. The first five characters of the BLOCK_ID gives the County FIPS code. The next six characters of the BLOCK_ID give the Tract FIPS code. The final four characters of the BLOCK_ID give the Block FIPS code. The school populations were summed across all county and tract combinations. Table E4a was calculated using the same methods as in the "Calculation

⁴ Ted Palma, EPA OAQPS, <u>palma.ted@epa.gov</u>, 919-541-5470.

- 1 of Measure" section, replacing counties by county / tract combinations, replacing county
- 2 populations of children 0 to 17 years by total school populations for each county / tract, and
 3 summing over all county / tract combinations in the schools database.
- 4

5 **Questions and Comments**

6

7 Questions regarding these methods, and suggestions to improve the description of the methods,

- 8 are welcome. Please use the "Contact Us" link at the bottom of any page in the America's
- 9 Children and the Environment website.
- 10
- 11
- 12